Amendments to the Claims

Please amend Claims 1, 2, and 3. Please cancel Claim 21. Please add claims 34-39. The Claim Listing below will replace all prior versions of the claims in the application:

Claim Listing

1.   (Currently Amended) A method of inhibiting TNFα in a human patient having inflammation associated with a-neurodegenerative disease, comprising administering to said human patient an effective TNFα-inhibiting amount of an anti-TNFα antibody or antigen-binding fragment thereof, said anti-TNFα antibody comprising a human constant region, wherein said anti-TNFα antibody or antigen binding fragment thereof (i) competitively inhibits binding of A2 (ATCC Accession No. PTA-7045) to human TNFα and (ii) binds to a neutralizing epitope of human TNFα with an affinity of at least 1×10^8 liter/mole, measured as an association constant (Ka), as determined by Scatchard analysis.

2.   (Currently Amended) A method of inhibiting TNFα in a human patient having inflammation associated with a-neurodegenerative disease, comprising administering to said human patient an effective TNFα-inhibiting amount of an anti-TNFα monoclonal antibody or antigen-binding fragment thereof, said anti-TNFα antibody comprising a human constant region, wherein said anti-TNFα chimeric antibody or antigen-binding fragment thereof (i) comprises the antigen-binding regions of A2 (ATCC Accession No. PTA-7045) and (ii) binds to a neutralizing epitope of human TNFα with an affinity of at least 1×10^8 liter/mole, measured as an association constant (Ka), as determined by Scatchard analysis.

3.   (Currently Amended) A method of inhibiting TNFα in a human patient having inflammation associated with a-neurodegenerative disease, comprising administering to said human patient an effective TNFα-inhibiting amount of an anti-TNFα antibody or antigen-binding fragment thereof, said anti-TNFα antibody comprising a human IgG1 constant region, wherein said anti-TNFα antibody or antigen-binding fragment thereof (i)

competitively inhibits the binding of A2 (ATCC Accession No. PTA-7045) to human

TNFα and (ii) binds to a neutralizing epitope of human TNFα with an affinity of at least 1

x 10⁸ liter/mole, measured as an association constant (Ka)~~, as determined by Scatchard

analysis~~.

4.   (Previously Presented) The method of Claim 3, wherein the anti-TNFα antibody

comprises a non-human variable region.

5.   (Previously Presented) The method of Claim 1, wherein said administration comprises a

single or divided 0.1 - 50 mg/kg dose of said anti-TNFα antibody or fragment thereof.

6.   (Previously Presented) The method of Claim 2, wherein said administration comprises a

single or divided 0.1 - 50 mg/kg dose of said anti-TNFα antibody or fragment thereof.

7.   (Previously Presented) The method of Claim 3, wherein said administration comprises a

single or divided 0.1 - 50 mg/kg dose of said anti-TNFα antibody or fragment thereof.

8. - 14. (Cancelled)

15.   (Previously Presented) The method of Claim 1 further comprising administering to the

human an effective amount of a pain control agent.

16.   (Previously Presented) The method of Claim 15, wherein the pain control agent is

selected from the group consisting of: paracetamol and dextropropoxyphene.

17.   (Cancelled).

18.   (Previously Presented) The method of Claim 1, wherein the anti-TNFα antibody is of

immunoglobulin class IgG1, IgG2, IgG3, IgG4 or IgM.

19. (Previously Presented) The method of Claim 1, wherein the anti-TNFα antibody is a fragment selected from the group consisting of Fab, Fab', F(ab')₂ and Fv.

20. (Previously Presented) The method of Claim 5 wherein said single or divided dose is one selected from 0.5, 0.9, 1, 1.1, 1.5, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14 or 15 mg/kg per day on at least one of day 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29 or 30 or at least one of week 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19 or 20.

21. (Cancelled).

22. (Previously Presented) The method of Claim 1, wherein the anti-TNFα antibody or antigen-binding fragment comprises a human constant region and a human variable region.

23. (Previously Presented) The method of Claim 1 wherein said anti-TNFα antibody or antigen-binding fragment comprises at least one human light chain and at least one human heavy chain.

24. (Previously Presented) The method of Claim 1, wherein said anti-TNFα antibody or antigen-binding fragment is administered to the human by means of parenteral administration.

25. (Previously Presented) The method of Claim 1, wherein said anti-TNFα antibody or antigen-binding fragment is administered to the human by means of intravenous administration, subcutaneous administration or intramuscular administration.

26. (Previously Presented) The method of Claim 23, wherein the light chain comprises all antigen-binding regions of the light chain of A2 (ATCC Accession No. PTA-7045).

27.   (Previously Presented) The method of Claim 23, wherein the heavy chain comprises all antigen-binding regions of the heavy chain of A2 (ATCC Accession No. PTA-7045).

28.   (Previously Presented) The method of Claim 23, wherein the light chain comprises all antigen-binding regions of the light chain of A2 (ATCC Accession No. PTA-7045) and the heavy chain comprises all antigen-binding regions of the heavy chain of A2 (ATCC Accession No. PTA-7045).

29.   (Previously Presented) The method of Claim 1, further comprising administering a composition comprising the anti-TNFα antibody or antigen-binding fragment of Claim 1 and a pharmaceutically acceptable carrier.

30.   (Previously Presented) The method of Claim 1, wherein said anti-TNFα antibody or antigen-binding fragment has specificity for a neutralizing epitope of human TNFα.

31.   (Previously Presented) The method of Claim 1, wherein said anti-TNFα antibody comprises a non-human variable region comprising an amino acid sequence selected from the group consisting of SEQ ID NO: 3 and SEQ ID NO: 5.

32.   (Previously Presented) The method of Claim 31, wherein the non-human variable region is murine.

33.   (Previously Presented) The method of Claim 32, wherein the non-human variable region comprises a polypeptide encoded by a nucleic acid sequence selected from the group consisting of SEQ ID NO: 2 and SEQ ID NO: 4.

34.   (New) A method of inhibiting TNFα in a human patient having inflammation associated with amylotrophic lateral sclerosis (ALS), comprising administering to said human patient an effective TNFα-inhibiting amount of an anti-TNFα antibody or antigen-binding fragment thereof, said anti-TNFα antibody comprising a human constant region, wherein said anti-TNFα antibody or antigen-binding fragment thereof (i) competitively inhibits

binding of A2 (ATCC Accession No. PTA-7045) to human TNFα and (ii) binds to a neutralizing epitope of human TNFα with an affinity of at least 1×10^8 liter/mole, measured as an association constant (Ka).

35. (New) A method of inhibiting TNFα in a human patient having inflammation associated with amylotrophic lateral sclerosis (ALS), comprising administering to said human patient an effective TNFα-inhibiting amount of an anti-TNFα antibody or antigen-binding fragment thereof, said anti-TNFα antibody comprising a human IgG1 constant region, wherein said anti-TNFα antibody or antigen-binding fragment thereof (i) competitively inhibits the binding of A2 (ATCC Accession No. PTA-7045) to human TNFα and (ii) binds to a neutralizing epitope of human TNFα with an affinity of at least 1×10^8 liter/mole, measured as an association constant (Ka).

36. (New) The method of Claim 34, wherein the anti-TNFα antibody comprises a non-human variable region.

37. (New) The method of Claim 34, wherein said administration comprises a single or divided 0.1 - 50 mg/kg dose of said anti-TNFα antibody or fragment thereof.

38. (New) The method of Claim 34 further comprising administering to the human an effective amount of a pain control agent.

39. (New) The method of Claim 38, wherein the pain control agent is selected from the group consisting of: paracetamol and dextropropoxyphene.